

reduction in Cornell product (-150 ± 849 vs -225 ± 816 mm \cdot msec, $p=.004$) and consequent greater prevalence of Cornell product LVH (56.0 vs 48.1% , $p<.001$), despite slightly greater decreases in systolic (-31.0 ± 20.0 vs -29.4 ± 19.5 mm Hg, $p=.011$) and diastolic BP (-17.8 ± 11.1 vs -17.0 ± 10.2 mm Hg, $p=.017$). After controlling for these baseline and in-study differences and for the differential treatment effect of losartan vs atenolol using analysis of covariance, diabetics had significantly less reduction in Cornell product (adjusted mean change -115 vs -226 mm \cdot msec, $p=0.009$) than nondiabetics, and, using multivariate logistic regression analysis, had a 1.31-fold greater risk of having ECG LVH by Cornell product at last study measurement (95% CI 1.12-1.52, $p<.001$).

Conclusion: Diabetic hypertensive patients have less regression of ECG LVH than nondiabetics. Further study will be needed to assess to what degree this accounts for their higher CVMM.

10:45 a.m.

890-2 Blood Pressure, Weight, and Cholesterol

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Background: To examine the blood pressure (BP), weight, and cholesterol data from the Program on the Surgical Control of the Hyperlipidemias (POSCH). Blood pressure (BP) reductions have been attributed to extra-lipid effects of statin drugs. In POSCH no statin drugs were employed and marked lipid reduction was achieved by the partial ileal bypass operation.

Methods: Plasma total and low-density lipoprotein (LDL) cholesterol, BP, and body weight (as body mass index, BMI) in POSCH patients at baseline and at 5-years post-randomization were compared.

Results: There were no statistically significant differences in mean systolic or diastolic BP between the control and intervention groups at baseline. At 5 years, however, statistically significant ($p<0.0005$) differences in mean systolic and diastolic BP were found between the groups. Similarly, the differences between the groups in the BMI and the plasma total and LDL-cholesterol were not statistically significant at baseline, but were at 5 years ($p<0.0001$). By multivariate regression analyses, consistent, statistically significant relationships were found between BMI and BP ($p<0.0001$). In addition, for systolic BP, in the entire POSCH population ($n=838$), a statistically significant relationship was found on regression analysis for the plasma total cholesterol ($p<0.013$).

Conclusions: POSCH is the only major lipid/atherosclerosis trial to establish a relationship between cholesterol reduction and BP reduction without the need to implicate statin drugs' extra-lipid effects.

11:00 a.m.

890-3 Adiposity-Independent Sympathetic Overactivity in African American Men

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Background: A large body of clinical investigation implicates an important role for the sympathetic nervous system in linking obesity with hypertension. However, most of the experimental support for this hypothesis has been derived from strictly white cohorts. We recently demonstrated, in a cross-sectional study, that sympathetic nerve activity (SNA) is closely correlated with body mass index (BMI) in Caucasians and in African American women, but, surprisingly, not in African American men. This dissociation is explained by a uniformly high sympathetic nerve firing rate in African American men, regardless of adiposity.

Methods: To further test this ethnic- and gender- specific dissociation, we now performed a prospective weight loss study comparing effects of hypocaloric diet on BMI and SNA in 11 obese African American men and 6 obese African American women. SNA is measured with microelectrodes inserted selectively into muscle nerve fascicles of the peroneal nerves, before and 16 weeks after hypocaloric diet.

Results: The major new finding is that, in normotensive obese African American men, the dietary-induced weight loss of 11.2 ± 0.9 kg (from BMI of 32.7 ± 1.2 to 29.0 ± 1.1) had no effect on SNA (from baseline of 26 ± 4 to 27 ± 3 bursts/min, $p = NS$). In contrast, in young African American women, even a smaller amount of weight loss of 9.1 ± 0.9 kg (from BMI of 33.2 ± 2.5 to 29.9 ± 2.7) was accompanied by reduction in SNA by more than 50% (from baseline of 22 ± 3 to 10 ± 1 bursts/min, $p = 0.02$). These differences were not explained by dietary sodium content or urinary sodium excretion.

Conclusion: These new data from our prospective study provide the strong support for a major adiposity-independent sympathetic overactivity in African American men and adiposity-related sympathetic overactivity in African American women. The data suggest a potential neurophysiologic explanation for the clinical observation that weight gain does not predict incident hypertension in African American men.

11:15 a.m.

890-4 Relation of the Metabolic Syndrome to Cardiac Markers of Preclinical Disease: The Strong Heart Study

Marcello Chinali, Richard B. Devereux, Barbara V. Howard, Mary J. Roman, Jonathan N. Bella, Jennifer E. Liu, Helaine E. Resnick, Elisa T. Lee, Lyle Best, Richard R. Fabsitz, Giovanni de Simone, Weill Medical College of Cornell University, New York, NY

Background. Metabolic syndrome (MS) is linked to cardiovascular risk. Recently published Adult Treatment Panel III (ATPIII) criteria provide a definition for diagnosis of MS. We analyzed the prevalence of cardiac markers of pre-clinical disease in MS participants and the impact of blood pressure on cardiac structure and function in the context of MS.

Methods and Results. Left ventricular (LV) structure and function were examined in 595 non-diabetic SHS participants (31.5% men) with MS and in 809 normal participants (59.5% men). Among participants with MS, subgroups with or without non-optimal blood pressure ($\geq 130/85$ mmHg) were compared. Participants with MS were older (60 vs 59 years, $p<0.05$) and more often women ($p<0.001$). After controlling these covariates, MS participants exhibited higher LV diastolic diameter, LV mass, relative wall thickness, aortic root and left atrial dimensions and higher prevalence of LV hypertrophy (all $p \leq 0.001$). Ejection fraction was reduced ($p \leq 0.01$) and LV relaxation (lower E/A ratio and higher mitral deceleration time) was prolonged (both $p \leq 0.05$). No differences were observed in BMI, body surface area, heart rate, plasma insulin or fasting glucose (all $p=ns$) between participants with the MS with or without non-optimal blood pressure. However, in the context of MS, non-optimal blood pressure was associated with higher LV mass and hypertrophy (both $p \leq 0.001$), and prolonged LV relaxation (lower E/A ratio and longer mitral deceleration time; both $p \leq 0.01$) whereas cardiac findings in normotensive participants with the MS were similar to those in the non-MS participants.

Conclusion. Abnormal LV geometry and function is related to MS by ATPIII definition only when blood pressure is elevated. Among participants with MS, those with non-optimal blood pressure have cardiac abnormalities associated with impaired prognosis.

11:30 a.m.

890-5 Systolic Blood Pressure Control in Diabetics and Nondiabetics With Hypertension and Coronary Artery Disease

Rhonda M. Cooper-DeHoff, Heather A. Bristol, Carl J. Pepine, University of Florida, Gainesville, FL

Objective: To characterize systolic blood pressure (SBP) control in a population of diabetic and nondiabetic patients with hypertension and coronary artery disease. **Methods:** An analysis was undertaken of 4,812 diabetics and 11,038 nondiabetics enrolled in the International Verapamil/trandolapril Study (INVEST) and treated for 1 year. INVEST compares strategies containing either verapamil SR or atenolol and/or trandolapril and hydrochlorothiazide, relative to BP control (JNC VI goal of $<130/85$ mmHg in diabetics and $<140/90$ mmHg in nondiabetics and adverse outcomes. **Results:** A total of 4,237 (88%) diabetics and 8,313 (75%) nondiabetics had SBP above the JNC VI goal at baseline despite antihypertensive treatment in 91% and 85%, respectively. Additionally, at baseline, the percentage of diabetics and nondiabetics with SBP (in mmHg) >169 , 160-169, 150-159 and 140-149 were all similar. SBP and strategy drug use at 12 months are characterized in the table. At 12 months, diabetic patients received 3 strategy drugs twice as often as nondiabetics, which resulted in SBP reductions to either <140 mmHg or <130 mmHg in similar percentages. **Conclusion:** Results indicate that after 12 months of treatment, 73% of all nondiabetics achieved JNC VI SBP goal of <140 mmHg, but only 43% of diabetics achieved JNC VI SBP goal of <130 mmHg. These results confirm that it is more difficult to control SBP in diabetics if JNC VI goals are used, despite the use of more strategy drugs.

	Diabetics (n=4,812)	Nondiabetics (n=11,038)
Baseline SBP above JNC goal n (%)	4,237 (88)	8,312 (75)
12 Mo SBP <140 mmHg n (%)	3,335 (69)	8,082 (73)
12 Mo SBP <130 mmHg n (%)	2,049 (43)	4,774 (43)
Receiving 2 strategy drugs n (%)	1,776 (37)	4,451 (40)
Receiving 3 strategy drugs n (%)	1,998 (42)	2,955 (27)

11:45 a.m.

890-6 Rofecoxib, but Not Celecoxib or Naproxen, Increases Mean 24-Hour Systolic Blood Pressure in Treated Hypertensive Patients With Osteoarthritis and Type 2 Diabetes Mellitus

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Background: Non-specific NSAIDs are known to destabilize blood pressure (BP) in treated hypertensive patients. BP destabilization is of particular concern in Type 2 diabetics, given their increased risk of CV disease. Therefore, we compared the effects of the COX-2 specific inhibitors celecoxib (C) and rofecoxib (R) and the NSAID naproxen in patients with ACE inhibitor-treated hypertension (HTN), Type 2 DM, and osteoarthritis (OA).

Methods: This was a multicenter, double-blind, randomized, 12 week study. Patients received C 200 mg QD ($n=136$), R 25 mg QD ($n=138$), or N 500 mg BID ($n=130$). 24 hour ambulatory BP measurements (ABPM) were performed at baseline, and after 6 and 12 weeks of therapy. The primary outcome measure was the mean change from baseline to week 6 of the average 24-hour systolic BP (SBP). Several validated arthritis efficacy assessments were performed at Weeks 6 and 12. A two-way ANOVA with investigational site and treatment as factors were used to assess the differences in BP among the 3 treatment groups.

Results: The mean age of patients was 63; 60% were female. Mean SBP levels at baseline were 132, 132, and 134 mmHg for the C, R, and N groups, respectively. At week 6, R induced a significant increase in 24-hour SBP ($+4.2$ mmHg), whereas C and N did not (-0.1 and -0.8 mmHg respectively; $P=.005$). Treatment differences were as follows: C vs R (95% CI) -3.8 (-6.4 to -1.2 ; $P=.005$); C vs N $+0.1$ (-2.6 to 2.76 ; $P=.956$); R vs N $+3.9$ (1.2 to 6.6 ; $P=.005$). Results for ambulatory SBP at Week 12 were comparable to Week 6. Comparative doses of these drugs were equally effective in improvement of OA: